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 NEWS 18  MAY 02   MEDLINE Improvements Provide Fast and Simple Access to DOI and
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 NEWS 19  MAY 12   European Patent Classification thesauri added to the INPADOC
                   files, PCTFULL, GBFULL and FRFULL
 NEWS 20  MAY 23   Enhanced performance of STN biosequence searches
 NEWS 21  MAY 23   Free Trial of the Numeric Property Search Feature
                   in PCTFULL on STN
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 NEWS 23  JUN 20   INPADOC databases enhanced with first page images
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 NEWS EXPRESS 17 DECEMBER 2010 CURRENT WINDOWS VERSION IS V8.4.2 .1,
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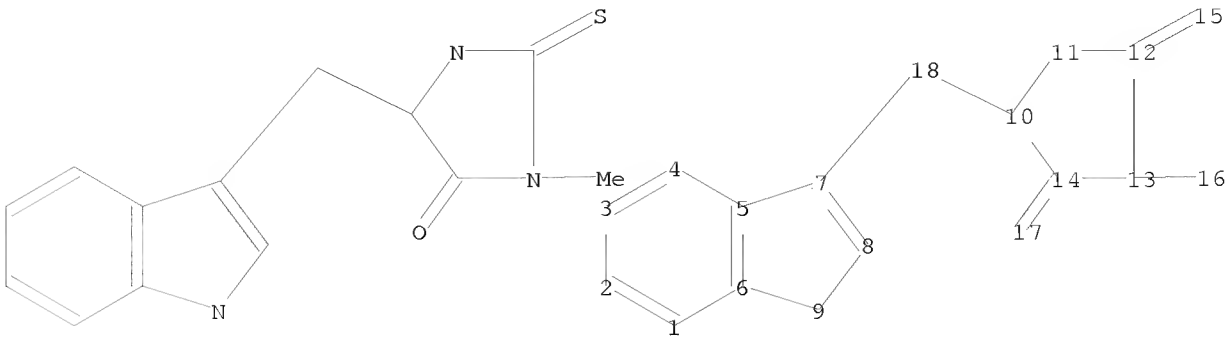

chain nodes :
15 16 17 18
ring nodes :
1 2 3 4 5 6 7 8 9 10 11 12 13 14
chain bonds :
7-18 10-18 12-15 13-16 14-17
ring bonds :
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exact/norm bonds :
5-7 6-9 7-8 8-9 10-11 10-14 11-12 12-13 12-15 13-14 14-17
exact bonds :
7-18 10-18 13-16
normalized bonds :
1-2 1-6 2-3 3-4 4-5 5-6

Match level :
1:Atom 2:Atom 3:Atom 4:Atom 5:Atom 6:Atom 7:Atom 8:Atom 9:Atom 10:Atom
11:Atom 12:Atom 13:Atom 14:Atom 15:CLASS 16:CLASS 17:CLASS 18:CLASS

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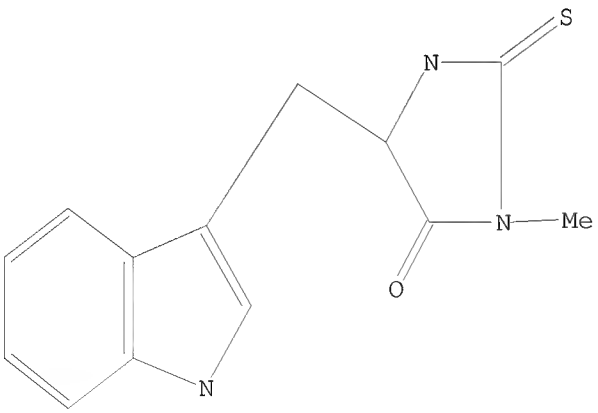
L1 HAS NO ANSWERS
L1 STR



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=> s l1 fam ful
FULL SEARCH INITIATED 14:49:06 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED - 116 TO ITERATE

100.0% PROCESSED 116 ITERATIONS 2 ANSWERS
SEARCH TIME: 00.00.01

L2 2 SEA FAM FUL L1

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FULL ESTIMATED COST 78.27 78.50

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FILE LAST UPDATED: 7 Jul 2011 (20110707/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Apr 2011
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Apr 2011

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=> s l2
L3 52 L2

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4 52 DUP REM L3 (0 DUPLICATES REMOVED)

=> s l4 and (cancer or tumor or neoplasm)
L5 52 S L4
516396 CANCER
75799 CANCERS
534736 CANCER
(CANCER OR CANCERS)
603368 TUMOR

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        211412 TUMORS
        667028 TUMOR
                  (TUMOR OR TUMORS)
        657655 NEOPLASM
         39830 NEOPLASMS
        675357 NEOPLASM
                  (NEOPLASM OR NEOPLASMS)
L6          17 L5 AND (CANCER OR TUMOR OR NEOPLASM)

=> d l6 ibib abs 1-17

L6   ANSWER 1 OF 17  CAPLUS  COPYRIGHT 2011 ACS on STN
ACCESSION NUMBER:        2011:13395  CAPLUS
DOCUMENT NUMBER:         154:580040
TITLE:                   Necrostatin decreases oxidative damage, inflammation,
                         and injury after neonatal HI
AUTHOR(S):               Northington, Frances J.; Chavez-Valdez, Raul; Graham,
                         Ernest M.; Razdan, Sheila; Gauda, Estelle B.; Martin,
                         Lee J.
CORPORATE SOURCE:        Neonatal Research Laboratory, Department of
                         Pediatrics, Johns Hopkins University School of
                         Medicine, Baltimore, MD, USA
SOURCE:                  Journal of Cerebral Blood Flow
& Metabolism (2011),
                         31(1), 178-189
                         CODEN: JCBMDN; ISSN: 0271-678X
PUBLISHER:               Nature Publishing Group
DOCUMENT TYPE:           Journal
LANGUAGE:                English
AB   Necrostatin-1 inhibits receptor-interacting protein (RIP)-1 kinase and
     programmed necrosis and is neuroprotective in adult rodent models.  Owing
     to the prominence of necrosis and continuum cell death in neonatal
     hypoxia-ischemia (HI), we tested whether necrostatin was neuroprotective
     in the developing brain.  Postnatal day (P)7 mice were exposed to HI and
     injected intracerebroventricularly with 0.1 μL of 80 μmol
     necrostatin, Nec-1, 5-(1H-Indol-3-ylmethyl)-(2-thio-3-methyl) hydantoin,
     or vehicle.  Necrostatin significantly decreased injury in the forebrain
     and thalamus at P11 and P28.  There was specific neuroprotection in
     necrostatin-treated males.  Necrostatin treatment decreased necrotic cell
     death and increased apoptotic cell death.  Hypoxia-ischemia enforced
     RIP1-RIP3 complex formation and inhibited RIP3-FADD (Fas-associated protein
     with death domain) interaction, and these effects were blocked by
     necrostatin.  Necrostatin also decreased HI-induced oxidative damage to
     proteins and attenuated markers of inflammation coincidental with
     decreased nuclear factor-κB and caspase 1 activation, and FLIP
     ((Fas-associated death-domain-like IL-1β-converting enzyme)-inhibitory
     protein) gene and protein expression.  In this model of severe neonatal
     brain injury, we find that cellular necrosis can be managed
     therapeutically by a single dose of necrostatin, administered after HI,
     possibly by interrupting RIP1-RIP3-driven oxidative injury and
     inflammation.  The effects of necrostatin treatment after HI reflect the
     importance of necrosis in the delayed phases of neonatal brain injury and
     represent a new direction for therapy of neonatal HI.  Journal of Cerebral
     Blood Flow & Metabolism (2011) 31, 178-189;
doi:10.1038/jcbfm.2010.72;
     published online 23 June 2010.
OS.CITING REF COUNT:     1     THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD
                               (1 CITINGS)
REFERENCE COUNT:         40    THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6   ANSWER 2 OF 17  CAPLUS  COPYRIGHT 2011 ACS on STN
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ACCESSION NUMBER: 2010:1519973 CAPLUS
DOCUMENT NUMBER: 155:9217
TITLE: Chemical probing reveals insights into the signaling mechanism of inflammasome activation
AUTHOR(S): Gong, Yi-Nan; Wang, Xiaoming; Wang, Jiayi; Yang, Zhenxiao; Li, Shan; Yang, Jieling; Liu, Liping; Lei, Xiaoguang; Shao, Feng
CORPORATE SOURCE: College of Life Sciences, Beijing Normal University, Beijing, 100875, Peop. Rep. China
SOURCE: Cell Research (2010), 20(12), 1289-1305
CODEN: CREEB6; ISSN: 1001-0602
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Caspase-1-mediated IL-1β production is generally controlled by two pathways. Toll-like receptors (TLRs) recognize pathogen-derived products and induce NF-κB-dependent pro-IL-1β transcription; NOD-like receptors (NLRs) assemble caspase-1-activating inflammasome complexes that sense bacterial products/danger signals. Through a targeted chemical screen, we identify bromoxone, a marine natural product, as a specific and potent inhibitor of the caspase-1 pathway. Bromoxone is effective over diverse inflammatory stimuli including TLR ligands plus ATP/nigericin, cytosolic DNA, flagellin and Bacillus anthracis lethal toxin. Bromoxone also efficiently suppresses caspase-1 activation triggered by several types of bacterial infection. Bromoxone acts upstream or at the level of the inflammasome in a transcription-independent manner. Bromoxone also inhibits pro-IL-1β expression by targeting components upstream of IKK in the TLR-NF-κB pathway. The unique dual activities of bromoxone are shared by the known TAK1 inhibitor that specifically blocks Nalp3 inflammasome activation. Hinted from the mechanistic and pharmacol. properties of bromoxone, we further discover that several known NF-κB inhibitors that act upstream of IKK, but not those targeting IKK or IKK downstream, are potent blockers of different NLRs-mediated caspase-1 activation. Our study uncovers a possible non-transcriptional mol. link between the NLR (Nalp3)-mediated inflammasome pathway and TLR-NF-κB signaling, and suggests a potential strategy to develop new anti-inflammatory drugs.
REFERENCE COUNT: 69 THERE ARE 69 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 3 OF 17 CAPLUS COPYRIGHT 2011 ACS on STN
ACCESSION NUMBER: 2010:208691 CAPLUS
TITLE: Methods to analyze cellular necroptosis
AUTHOR(S): Miao, Benchun; Degterev, Alexei
CORPORATE SOURCE: Department of Biochemistry, Tufts University School of Medicine, Boston, MA, USA
SOURCE: Methods in Molecular Biology (Totowa, NJ, United States) (2009), 559(Apoptosis), 79-93
CODEN: MMBIED; ISSN: 1064-3745
PUBLISHER: Humana Press Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Necroptosis is a mechanism of necrotic cell death induced by external stimuli in the form of death domain receptor (DR) engagement by their resp. ligands, TNF-alpha, Fas ligand (FasL) and TRAIL, under conditions when apoptotic cell death execution is prevented, e.g. by caspase inhibitors. Although it occurs under regulated conditions, necroptotic cell death is characterized by the same morphol. features as unregulated necrotic death. RIP1 kinase activity is a key step in the necroptosis pathway. We have previously identified specific and potent small-mol. inhibitors of necroptosis, necrostatins, which efficiently prevent execution of this form of cell death. Herein, we describe the methods to

analyze cellular necroptosis, and the methods to analyze the inhibitory effects of anti-necroptosis compds. (necrostatin-1).

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 4 OF 17 CAPLUS COPYRIGHT 2011 ACS on STN

ACCESSION NUMBER: 2010:81469 CAPLUS

DOCUMENT NUMBER: 152:184285

TITLE: Tryptophan catabolism in cancer treatment and diagnosis

INVENTOR(S): Van Den Eynde, Benoit; Pilotte, Luc; De Plaen, Etienne

PATENT ASSIGNEE(S): Ludwig Institute for Cancer Research Ltd., USA

SOURCE: PCT Int. Appl., 74pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2010008427	A1	20100121	WO 2009-US2250	20090410
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
US 20110159017	A1	20110630	US 2011-936576	20110302

PRIORITY APPLN. INFO.:			
US 2008-123940P	P	20080411	
WO 2009-US2250	W	20090410	

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The unexpected expression of tryptophan 2,3-dioxygenase (TDO2)in cancer cells and tumors has been established. Methods for diagnosing cancer based on the expression of TDO2 are provided, as are methods for treating cancer and inhibiting the growth of cancer cells by inhibiting TDO2, as well as pharmaceutical compns.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 5 OF 17 CAPLUS COPYRIGHT 2011 ACS on STN

ACCESSION NUMBER: 2008:1539661 CAPLUS

DOCUMENT NUMBER: 150:391271

TITLE: Participation of necroptosis in neuroblastoma cell death induced by aluminum

AUTHOR(S): Zhang, Qinli; Niu, Qiao; Zhang, Ling; Wang, Liang

CORPORATE SOURCE: Department of Occupational Health, School of Public Health, Shanxi Medical University, Taiyuan, 030001, Peop. Rep. China

SOURCE: Zhongguo Yaolixue Yu Dulixue Zazhi (2008), 22(5), 382-390
CODEN: ZYYZEW; ISSN: 1000-3002

PUBLISHER: Zhongguo Yaolixue Yu Dulixue Zazhi Biarjibu

DOCUMENT TYPE: Journal

LANGUAGE: Chinese

AB The role of necroptosis in the mechanisms of aluminum-induced cell death was investigated. The aluminum-induced in vitro cell death model was prepared by treating SH-SY5Y cells with AlCl3/6H2O at 4 mmol/L-1, and RNA interference (RNAi) was performed to suppress the expression of caspase 3 gene, and necrostatin-1 (Nec-1) was added into culture to restrain necroptosis. Cell viability was detected under different treatments and at diverse time courses after treatment. Interference efficiency was measured by QRT-PCR, and apoptosis rate and necrotic rate were measured with cytometry. Finally, level of protein expression was quantified with immunohistochem. Based on the viabilities in different caspase 3 RNA small interference sequences, transfection concentration and transfection course, the optimal transfection concentration was determined as 10 nmol/L-1, and the optimal transfection time was 48 h after transfection. Furthermore, caspase 3 siRNA 1 was selected as the most effective sequence according to the gene expression of different siRNA sequences, and the transfection efficiency was 93.0%, and the interference efficiency was 63.0%, and there was also a significant difference in the expression of caspase 3. The cell viability could be improved significantly by caspase 3 siRNA, and the apoptotic rate was reduced simultaneously. On the other hand, the viability could be enhanced significantly by Nec-1, and the necrotic rate was decreased. Cooperation of caspase 3 siRNA 1 and Nec-1 could increase the cell viability significantly, and both apoptotic rate and necrotic rate were enhanced. In summary necroptosis was present in the cell death pathway of aluminum induced cell death besides apoptosis and necrosis.

L6 ANSWER 6 OF 17 CAPLUS COPYRIGHT 2011 ACS on STN

ACCESSION NUMBER: 2008:1136027 CAPLUS

DOCUMENT NUMBER: 149:462087

TITLE: Structure-activity relationship study of a novel necroptosis inhibitor, necrostatin-7

AUTHOR(S): Zheng, Weihong; Degterev, Alexei; Hsu, Emily; Yuan, Junying; Yuan, Chengye

CORPORATE SOURCE: State Key Laboratory of Bio-Organic and Natural Product Chemistry, Shanghai Institute of Organic Chemistry, Chinese Academy of Sciences, Shanghai, 200032, Peop. Rep. China

SOURCE: Bioorganic & Medicinal Chemistry Letters (2008), 18(18), 4932-4935
CODEN: BMCLE8; ISSN: 0960-894X

PUBLISHER: Elsevier Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

OTHER SOURCE(S): CASREACT 149:462087

AB Necroptosis is a regulated caspase-independent cell death mechanism characterized by morphol. features resembling non-regulated necrosis. Necrotatin-7 (Nec-7), a novel potent small-mol. inhibitor of necroptosis, is structurally distinct from previously described necrostatins (Nec-1, Nec-3, Nec-4 and Nec-5). Here, we describe a series of structural modifications and the structure-activity relationship (SAR) of the Nec-7 series for inhibiting necroptosis.

OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)

REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 7 OF 17 CAPLUS COPYRIGHT 2011 ACS on STN

ACCESSION NUMBER: 2008:1021408 CAPLUS

DOCUMENT NUMBER: 150:206161

TITLE: Necrostatin-1 reduces histopathology and improves functional outcome after controlled cortical impact in mice
AUTHOR(S): You, Zerong; Savitz, Sean I.; Yang, Jinsheng; Degterev, Alexei; Yuan, Junying; Cuny, Gregory D.; Moskowitz, Michael A.; Whalen, Michael J.
CORPORATE SOURCE: Neuroscience Center, Massachusetts General Hospital, Harvard Medical School, Charlestown, MA, 02129, USA
SOURCE: Journal of Cerebral Blood Flow & Metabolism (2008), 28(9), 1564-1573
CODEN: JCBMDN; ISSN: 0271-678X
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Necroptosis is a newly identified type of programmed necrosis initiated by the activation of tumor necrosis factor alpha (TNFα)/Fas. Necrostatin-1 is a specific inhibitor of necroptosis that reduces ischemic tissue damage in exptl. stroke models. We previously reported decreased tissue damage and improved functional outcome after controlled cortical impact (CCI) in mice deficient in TNFα and Fas. Hence, we hypothesized that necrostatin-1 would reduce histopathol. and improve functional outcome after CCI in mice. Compared with vehicle-/inactive analog-treated controls, mice administered necrostatin-1 before CCI had decreased propidium iodide-pos. cells in the injured cortex and dentate gyrus (6 h), decreased brain tissue damage (days 14, 35), improved motor (days 1 to 7), and Morris water maze performance (days 8 to 14) after CCI. Improved spatial memory was observed even when drug was administered 15 mins after CCI. Necrostatin-1 treatment did not reduce caspase-3-pos. cells in the dentate gyrus or cortex, consistent with a known caspase-independent mechanism of necrostatin-1. However, necrostatin-1 reduced brain neutrophil influx and microglial activation at 48 h, suggesting a novel anti-inflammatory effect in traumatic brain injury (TBI). The data suggest that necroptosis plays a significant role in the pathogenesis of cell death and functional outcome after TBI and that necrostatin-1 may have therapeutic potential for patients with TBI. Journal of Cerebral Blood Flow & Metabolism (2008) 28, 1564-1573; doi:10.1038/jcbfm.2008.44; published online 21 May 2008.
OS.CITING REF COUNT: 13 THERE ARE 13 CAPLUS RECORDS THAT CITE THIS RECORD (13 CITINGS)
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 8 OF 17 CAPLUS COPYRIGHT 2011 ACS on STN
ACCESSION NUMBER: 2008:530303 CAPLUS
DOCUMENT NUMBER: 149:69718
TITLE: A key in vivo antitumor mechanism of action of natural product-based brassinins is inhibition of indoleamine 2,3-dioxygenase
AUTHOR(S): Banerjee, T.; DuHadaway, J. B.; Gaspari, P.; Sutanto-Ward, E.; Munn, D. H.; Mellor, A. L.; Malachowski, W. P.; Prendergast, G. C.; Muller, A. J.
CORPORATE SOURCE: NewLink Genetics Corporation, Ames, IA, USA
SOURCE: Oncogene (2008), 27(20), 2851-2857
CODEN: ONCNES; ISSN: 0950-9232
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Agents that interfere with tumoral immune tolerance may be useful to prevent or treat cancer. Brassinin is a phytoalexin, a class of natural products derived from plants that includes the widely known compound

resveratrol. Brassinin has been demonstrated to have chemopreventive activity in preclin. models but the mechanisms underlying its anticancer properties are unknown. Here, we show that brassinin and a synthetic derivative 5-bromo-brassinin (5-Br-brassinin) are bioavailable inhibitors of indoleamine 2,3-dioxygenase (IDO), a pro-tolerogenic enzyme that drives immune escape in cancer. Like other known IDO inhibitors, both of these compds. combined with chemotherapy to elicit regression of autochthonous mammary gland tumors in MMTV-Neu mice. Furthermore, growth of highly aggressive melanoma isograft tumors was suppressed by single agent treatment with 5-Br-brassinin. This response to treatment was lost in athymic mice, indicating a requirement for active host T-cell immunity, and in IDO-null knockout mice, providing direct genetic evidence that IDO inhibition is essential to the antitumor mechanism of action of 5-Br-brassinin. The natural product brassinin thus provides the structural basis for a new class of compds. with in vivo anticancer activity that is mediated through the inhibition of IDO.

OS.CITING REF COUNT: 27 THERE ARE 27 CAPLUS RECORDS THAT CITE THIS RECORD (28 CITINGS)

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 9 OF 17 CAPLUS COPYRIGHT 2011 ACS on STN

ACCESSION NUMBER: 2008:421553 CAPLUS

DOCUMENT NUMBER: 149:298787

TITLE: Down-regulation of the indoleamine 2, 3-dioxygenase (IDO) transcription by tryptophan analogues

AUTHOR(S): Okamoto, Takeaki; Tone, Shigenobu; Kanoichi, Hiroaki; Ohyama, Fumio; Minatogawa, Yohsuke

CORPORATE SOURCE: Department of Biochemistry, Kawasaki Medical School, 577 Matsushima, Kurashiki, Okayama, 701-0192, Japan

SOURCE: International Congress Series (2007), 1304(Interdisciplinary Conference on Tryptophan and Related Substances: Chemistry, Biology, and Medicine, 2006), 352-356
CODEN: EXMDA4; ISSN: 0531-5131

PUBLISHER: Elsevier B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Indoleamine 2,3-dioxygenase (IDO; EC 1.13.11.42) is a rate-limiting enzyme involved in the catabolism of tryptophan, which is an essential amino acid. It is induced under pathol. conditions, such as the presence of viral infections or tumor cells. This enzyme is induced by IFN-γ in the mouse rectal carcinoma cell line CMT-93. It is known that both 1-methyl-L-tryptophan (1-MT) and methylthiohydantoin-DL-tryptophan (MTH-trp) are tryptophan analogs, and are authentic inhibitors of the enzymic activity of IDO. In this study, we examined the effects of both 1-MT and MTH-trp on the IFN-γ inducible IDO expression of CMT-93. As a result, the IFN-γ inducible IDO mRNA and the protein levels in CMT-93 were suppressed by 1-MT and MTH-trp, independently. Moreover, tryptophan (Trp), as a substrate of IDO, also suppressed IDO induction by IFN-γ at the transcriptional level. These results suggest that 1-MT and MTH-trp as inhibitors of IDO enzymic activity, and Trp suppress IDO induction by IFN-γ at the transcriptional level.

REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 10 OF 17 CAPLUS COPYRIGHT 2011 ACS on STN

ACCESSION NUMBER: 2007:830612 CAPLUS

DOCUMENT NUMBER: 148:282740

TITLE: Transcriptional regulation of indoleamine 2,3-dioxygenase (IDO) by tryptophan and its analogue

AUTHOR(S): Okamoto, Takeaki; Tone, Shigenobu; Kanouchi, Hiroaki;

Miyawaki, Chie; Ono, Sayuri; Minatogawa, Yohsuke
CORPORATE SOURCE: Department of Biochemistry, Kawasaki Medical School, 577 Matsushima, Kurashiki, Okayama, 701-0192, Japan
SOURCE: Cytotechnology (2007), 54(2), 107-113
CODEN: CYTOER; ISSN: 0920-9069
PUBLISHER: Springer
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Indoleamine 2,3-dioxygenase (IDO; EC 1.13.11.42) is a rate-limiting enzyme involved in the catabolism of tryptophan, which is an essential amino acid. It is induced under pathol. conditions, such as the presence of viral infections or tumor cells. This enzyme is induced by IFN-γ in the mouse rectal carcinoma cell line CMT-93. It is known that both 1-methyl-l-tryptophan (1-MT) and methylthiohydantoin-dl-tryptophan (MTH-trp) are tryptophan analogs, and are authentic inhibitors of the enzymic activity of IDO. In this study, we examined the effects of both 1-MT and MTH-trp on the IFN-γ inducible IDO expression of CMT-93. As a result, the IFN-γ inducible IDO mRNA and the protein levels in CMT-93 were suppressed by 1-MT and MTH-trp, independently. Moreover, tryptophan (Trp), as a substrate of IDO, also suppressed IDO induction by IFN-γ at the transcriptional level. These results suggest that 1-MT and MTH-trp are as inhibitors of IDO enzymic activity, and Trp suppresses IDO induction by IFN-γ at the transcriptional level.

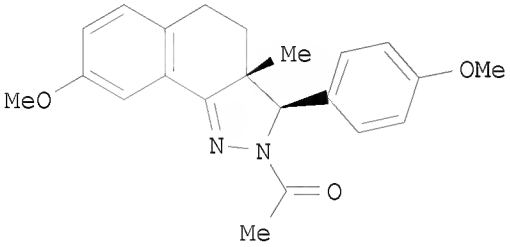
OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)
REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 11 OF 17 CAPLUS COPYRIGHT 2011 ACS on STN
ACCESSION NUMBER: 2007:730236 CAPLUS
DOCUMENT NUMBER: 147:143418
TITLE: Benzo[g]indazole, indole and tetralone compounds and their preparation, screening, and methods of treatment of diseases caused by TNFα or RIP1 protein
INVENTOR(S): Yuan, Junying; Degterev, Alexei; Hitomi, Junichi; Cuny, Gregory D.; Jagtap, Prakash
PATENT ASSIGNEE(S): President and Fellows of Harvard College, USA; The Brigham and Women's Hospital, Inc.
SOURCE: PCT Int. Appl., 263pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007075772	A2	20070705	WO 2006-US48583	20061220
WO 2007075772	A3	20090219		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
AU 2006331754	A1	20070705	AU 2006-331754	20061220

AU 2006331754	A2	20080814			
CA 2633500	A1	20070705	CA 2006-2633500		20061220
EP 1968583	A2	20080917	EP 2006-847822		20061220
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, RS					
JP 2009521454	T	20090604	JP 2008-547482		20061220
ZA 2008005748	A	20091230	ZA 2008-5748		20061220
IN 2008CN03693	A	20090313	IN 2008-CN3693		20080717
CN 101674826	A	20100317	CN 2006-80053077		20080820
US 20100190836	A1	20100729	US 2009-86792		20090622
PRIORITY APPLN. INFO.:			US 2005-751913P	P	20051220
			US 2006-843304P	P	20060908
			WO 2006-US48583	W	20061220

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): CASREACT 147:143418
GI



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AB The invention features compds., pharmaceutical compns., and methods for treating trauma, ischemia, stroke, degenerative diseases associated with cellular necrosis, and other conditions. Screening assays for identifying compds. useful for treating these conditions are also described. Example compound I was prepared by a multistep procedure (procedure given). All the invention compds. were evaluated for their necrosis inhibitory activity and their structure-activity relationship.
OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)

L6 ANSWER 12 OF 17 CAPLUS COPYRIGHT 2011 ACS on STN
ACCESSION NUMBER: 2007:337477 CAPLUS
DOCUMENT NUMBER: 146:408284
TITLE: Application of alkannin to prepare medicine inducing cytoclasis programmed death
INVENTOR(S): Hu, Xun; Han, Weidong
PATENT ASSIGNEE(S): Zhejiang University, Peop. Rep. China
SOURCE: Faming Zhuanli Shenqing, 20pp.
CODEN: CNXXEV
DOCUMENT TYPE: Patent
LANGUAGE: Chinese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CN 1931152	A	20070321	CN 2006-10053627	20060927
PRIORITY APPLN. INFO.:			CN 2006-10053627	20060927

AB The patent relates to application of

alkannin((+)-5,8-dihydroxy-2-(1-hydroxy-4-methyl-3-pentenyl)-1,4-naphthoquinone) to prepare medicine(liquid prepns., granules, tablets, medicinal instant granules, gelatin pills, capsules, sustained-release preparation, dripping pills or injections) inducing cytoclasis programmed death, and the medicine is composed of alkannin and medical excipient or carrier. The alkannin can kill multidrug resistance tumor cells, and has low toxicity.

L6 ANSWER 13 OF 17 CAPLUS COPYRIGHT 2011 ACS on STN
ACCESSION NUMBER: 2007:157223 CAPLUS
DOCUMENT NUMBER: 147:65087
TITLE: Chemical genetic approaches to probing cell death
AUTHOR(S): Gangadhar, Nidhi M.; Stockwell, Brent R.
CORPORATE SOURCE: Department of Biological Sciences, 614 Fairchild Center, New York, NY, 10027, USA
SOURCE: Current Opinion in Chemical Biology (2007), 11(1), 83-87
CODEN: COCBF4; ISSN: 1367-5931
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Chemical genetics has arisen as a tool for the discovery of pathways and proteins in mammalian systems. This approach, comprising small-mol. screening combined with biochem. and genomic target identification methods, enables one to assess which proteins are involved in regulating a particular phenotype. Applied to cell death, this strategy can reveal novel targets and pathways regulating the demise of mammalian cells. Numerous diseases have been linked to the loss of regulation of cell death. Defining the mechanisms governing cell death in these diseases might lead to the discovery of therapeutic agents and targets and provide a richer understanding of the mortality of living systems. Recent advances include the discovery of novel small mols. regulating cell death pathways - necrostatin and erastin - as well as the elucidation of the mechanism of death induced in cancer cells by the cytotoxic agent Apratoxin A.
OS.CITING REF COUNT: 9 THERE ARE 9 CAPLUS RECORDS THAT CITE THIS RECORD (9 CITINGS)
REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 14 OF 17 CAPLUS COPYRIGHT 2011 ACS on STN
ACCESSION NUMBER: 2005:369265 CAPLUS
DOCUMENT NUMBER: 142:423892
TITLE: Alanyl aminopeptidase inhibitors for functionally influencing different cells and treating immunological, inflammatory, neuronal, and other diseases
INVENTOR(S): Ansorge, Siegfried; Bank, Ute; Nordhoff, Karsten; Tager, Michael; Striggow, Frank
PATENT ASSIGNEE(S): Institut Fur Medizintechnologie Magdeburg GmbH IMTM, Germany; Keyneurotek AG
SOURCE: PCT Int. Appl., 332 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005037257	A2	20050428	WO 2004-EP11643	20041015
WO 2005037257	A3	20060914		

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RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

DE 10348023	A1	20050519	DE 2003-10348023		20031015
AU 2004281536	A1	20050428	AU 2004-281536		20041015
AU 2004281536	B2	20090709			
AU 2004281536	B9	20091008			
CA 2542723	A1	20050428	CA 2004-2542723		20041015
EP 1673075	A2	20060628	EP 2004-790485		20041015
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR					
CN 1897928	A	20070117	CN 2004-80036456		20041015
JP 2007508349	T	20070405	JP 2006-534706		20041015
US 20070037752	A1	20070215	US 2006-575882		20060915
PRIORITY APPLN. INFO.:			DE 2003-10348023	A	20031015
			WO 2004-EP11643	W	20041015

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): MARPAT 142:423892

AB The invention discloses substances which specifically inhibit peptidases splitting ala-p-nitroanilide for use in medicine. The invention further discloses the use of at least one such substance or at least one pharmaceutical or cosmetic composition containing such a substance for preventing and treating diseases, especially diseases with an overshooting immune response (autoimmune diseases, allergies, and transplant rejections), other chronic inflammatory diseases, neuronal diseases, brain damage, skin diseases (acne and psoriasis, among others), tumors, and special viral infections (including SARS).

OS.CITING REF COUNT: 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (10 CITINGS)
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 15 OF 17 CAPLUS COPYRIGHT 2011 ACS on STN
ACCESSION NUMBER: 2004:927197 CAPLUS
DOCUMENT NUMBER: 141:388648
TITLE: Novel ido (indoleamine 2,3-dioxygenase) inhibitors and methods of use
INVENTOR(S): Prendergast, George C.; Muller, Alexander J.; Duhadaway, James B.; Malachowski, William
PATENT ASSIGNEE(S): Lankenau Institute for Medical Research, USA
SOURCE: PCT Int. Appl., 115 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004094409	A1	20041104	WO 2004-US5154	20040220
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,				

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NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,
BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK,
TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2520586	A1	20041104	CA 2004-2520586	20040220
CA 2520586	C	20110614		
EP 1606285	A1	20051221	EP 2004-713430	20040220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1795187	A	20060628	CN 2004-80008331	20040220
CN 1794986	A	20060628	CN 2004-80014321	20040220
JP 2006521377	T	20060921	JP 2006-508788	20040220
CN 101265254	A	20080917	CN 2008-10092243	20040220
CN 101265259	A	20080917	CN 2008-10092244	20040220
EP 2260846	A1	20101215	EP 2010-75396	20040220
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US 20070173524	A1	20070726	US 2006-550444	20060601
US 7714139	B2	20100511		
US 20100233166	A1	20100916	US 2010-759066	20100413

PRIORITY APPLN. INFO.:	US 2003-458162P	P 20030327
	US 2003-527449P	P 20031205
	CN 2004-80008331	A3 20040220
	EP 2004-713378	A3 20040220
	WO 2004-US5154	W 20040220
	US 2006-550444	A1 20060601

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): MARPAT 141:388648

AB Novel inhibitors of indoleamine 2,3-dioxygenase (IDO) activity are provided. In yet another embodiment of the present invention, a combination treatment protocol comprising administration of an IDO inhibitor with a signal transduction inhibitor (STI) or chemotherapeutic agent is provided, which is effective for suppressing tumor growth. In still another embodiment of the present invention, a combination treatment protocol is provided for the treatment of a chronic viral infection, comprising the administration of an IDO inhibitor and a chemotherapeutic agent.

OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 16 OF 17 CAPLUS COPYRIGHT 2011 ACS on STN
ACCESSION NUMBER: 2004:927043 CAPLUS
DOCUMENT NUMBER: 141:388646
TITLE: Novel methods for the treatment of cancer and viral infections
INVENTOR(S): Prendergast, George C.; Muller, Alexander J.; Duhadaway, James B.; Malachowski, William
PATENT ASSIGNEE(S): Lankenau Institute for Medical Research, USA
SOURCE: PCT Int. Appl., 65 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 2004093871		A1	20041104	WO 2004-US5155	20040220
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	RW:	BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
CA 2520172		A1	20041104	CA 2004-2520172	20040220
EP 1613308		A1	20060111	EP 2004-713378	20040220
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CN 1795187		A	20060628	CN 2004-80008331	20040220
CN 1794986		A	20060628	CN 2004-80014321	20040220
JP 2006521378		T	20060921	JP 2006-508789	20040220
CN 101265254		A	20080917	CN 2008-10092243	20040220
CN 101265259		A	20080917	CN 2008-10092244	20040220
EP 2260846		A1	20101215	EP 2010-75396	20040220
	R:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LI, LU, MC, NL, PT, RO, SE, SI, SK, TR			
US 20070099844		A1	20070503	US 2006-551151	20060518
PRIORITY APPLN. INFO.:				US 2003-458162P	P 20030327
				US 2003-527449P	P 20031205
				CN 2004-80008331	A3 20040220
				EP 2004-713378	A3 20040220
				WO 2004-US5155	W 20040220

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Compns. and methods for the treatment of malignancy and chronic viral infection are disclosed. A method is claimed for treating a cancer comprising administering at least one indoleamine 2,3-dioxygenase (IDO) inhibitor and at least one signal transduction inhibitor (STI). A method is claimed for treating a cancer comprising administering at least one immunomodulator, other than IDO inhibitor, and at least one cytotoxic chemotherapeutic agent or at least one STI. A method for treating a chronic viral infection in a patient is claimed comprising administering at least one IDO inhibitor and at least one chemotherapeutic agent. Pharmaceutical compns. containing compds. of the invention for treating cancer and viral infections are also claimed.

OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 17 OF 17 CAPLUS COPYRIGHT 2011 ACS on STN

ACCESSION NUMBER: 2001:300459 CAPLUS

DOCUMENT NUMBER: 134:320879

TITLE: Small molecule inhibitors of necrosis

INVENTOR(S): Yuan, Junying; Degterev, Alexei; Mitchison, Timothy

PATENT ASSIGNEE(S): President and Fellows of Harvard College, USA

SOURCE: PCT Int. Appl., 68 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001028493	A2	20010426	WO 2000-US28475	20001013

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     WO 2001028493          A3    20010607
         W:  CA, JP
         RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE
     US 6756394             B1    20040629    US 2000-688015          20001013
     US 20050131044         A1    20050616    US 2004-880377          20040629
     US 7253201             B2    20070807
PRIORITY APPLN. INFO.:                         US 1999-159668P     P   19991015
                                               US 2000-174749P     P   20000106
                                               US 2000-688015      A1  20001013
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S):         MARPAT 134:320879
AB   The invention features methods for decreasing necrosis.  The invention
     also features methods for treating a subject with a condition in which
     necrosis occurs.  The invention further features chemical compds. used to
     decrease necrosis.
OS.CITING REF COUNT:     3     THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD
                               (4 CITINGS)
REFERENCE COUNT:         1     THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT


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                                                      ENTRY    SESSION
FULL ESTIMATED COST                                   63.04     141.54

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                      ENTRY    SESSION
CA SUBSCRIBER PRICE                                  -14.79     -14.79

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